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EDITORIAL COMMENT

Of That Waltz in My Heart*†

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The intrinsic anatomy of the left ventricular (LV) wall has fascinated anatomists over the centuries and resulted in varying designations ranging from descending and ascending bands, fascicles, and laminated layers to the more recent description as right- and left-handed helical arrangements of the subendocardial and subepicardial regions (1–4). In the looped adult heart, both inflow and outflow regions at the base of the LV are sinuously curved, lying side by side at the top of the LV. Near the apex, a well-formed loop of muscle fibers forms a spiral vortex, also referred to as "vortex cordis." Shortening of this counterdirectional mantle of muscle fibers results in a wringing movement of the LV that propels blood while the ensuing recoil initiates diastolic suction of blood into the LV cavity. It is therefore logical that the sequence of mechanical activation should aim for an apex-to-base acceleration of blood flow during systole and base-to-apex reversal during diastole. The studies by Sengupta et al. (5) and Ashikaga et al. (6) in this issue of the *Journal* put into perspective the flow sequences and the underlying electromechanical transients that modulate the cyclical switch between the dual roles of the vortex cordis in ventricular ejection and suction.

See pages 899 and 909

In the Wake of a Vortex

The passage of blood through the cardiac valves creates vortices similar to those generated by water in a narrow channel. An early observation linking natural vortex formation with the functioning of the cardiovascular system is found in a notebook of the Renaissance painter Leonardo da Vinci (7). In a well-designed investigation of the valve function, he modeled the aortic valve and sinuses of Valsalva from a cast of an oxen heart. Observing the vortices formed in the sinuses, he predicted the mechanism of closure of the aortic valves. As blood was forced through the valve, vortices in the sinuses edged back into the cusps of the valve. When the flow ceased, these vortices pushed the cusps against each other, forming a perfect seal to prevent reflux (7).

Modern-day investigations have confirmed several of da Vinci's observations. The streams of blood entering the right atrium from the superior and inferior cava do not collide, but move forward to form a vortex (1). In the left atrium, 2 temporally discrete crescent-shaped vortices are formed with an axis of rotation parallel to the mitral annular plane (8). With opening of the atrioventricular valves, the blood flow surges into the relaxed ventricles, again rotating and redirecting the flow toward the pulmonary artery and aorta, respectively. Previously, the sinuous curvature of the cardiac chambers was believed to determine the direction of blood flow toward ventricular outflow tracts (1). For example, the looped configuration of the adult LV and the geometry of the mitral valve lead to formation of a predominantly anteriorly directed vortex (1). It has not, however, been clear whether the sequence of LV depolarization and repolarization played a role in the redirection of blood flow. This was largely related to the limited temporal resolution of imaging techniques to visualize flow fields during isovolumic intervals.

The LV forces closing the mitral valve at the peak of electrocardiographic R-wave are generated before the entire ventricle is activated. The earliest electric activation in the LV occurs at the anteroseptal region and then travels in apex-to-base direction (9). The basal posterior wall is the last region to be activated near the end of the R-wave (9). Sengupta et al. (5) employed a high-temporal-resolution method to track the features of LV intracavitary blood flow in 2 dimensions during the brief isovolumic intervals. These investigators provided evidence that active flow acceleration in the apex-to-base direction joins and accentuates a wake vortex forming across the edge of a closing anterior mitral leaflet. These fluidic features may arguably regulate the time required for the cyclical change in LV contraction/filling, because a loss of well-directed flow and vortex formation was shown to delay closure of mitral valve and opening of aortic leaflets. It seems that the geometry of the heart, the pattern of its fibers, the blood flow within it, and the electromechanical activation sequence are intimately related. With contemplation, one begins to comprehend the heart's dynamic form and function, which the English anatomist J. Bell Pettigrew described as "exceedingly simple in principle but wonderfully complicated in detail" (3).

The Triple Beat of Waltz

The sequential contractile activity of the helicoid structure of the ventricular myocardium modulates the pattern of global LV motion observed in vivo. In previous experi-

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†From the London musical "The Dancing Years" (1939), Ivor Novello and Christopher Hassall.

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JACC Vol. 49, No. 8, 2007
February 27, 2007:917–20

ments, Sengupta et al. (10,11) compared the activation-contraction sequence of the subendocardial and subepicardial regions of porcine LV using sonomicrometry. Isovolumic contraction was characterized with greater shortening of the subendocardial fibers and was accompanied with stretching of the subepicardial fibers (10). This asynchronous deformation temporally coincided with biphasic isovolumic waves routinely recorded on tissue Doppler imaging (10,12). In this issue of the *Journal*, Ashikaga et al. (6) have extended these observations further and provided comprehensive assessment regarding the depth-dependent differences in myofiber mechanics in vivo. Their observations confirm the existence of a reciprocal relationship between the fiber and cross-fiber strains at epicardial and endocardial layers (6,10). During the isovolumic periods, fiber and cross-fiber strains in the same layer are in opposite directions, such that the geometric configuration of the LV wall changes without an alteration in LV volume. In another experimental study, Sengupta et al. (11) compared the shortening strains from the apex, mid, and basal regions of the LV and reported the presence of an apex-to-base gradient of longitudinal shortening, a finding that has been confirmed by Buckberg et al. (13). The intracavitary flow during the cardiac cycle described by Sengupta et al. (5) offers a vital rheologic rationale for the complex set of mechanical activity observed in vivo. Brief asynchronous deformation during the onset of systole and diastole may be advantageous in redirecting blood flow. For example, during isovolumic contraction, blood flow accelerates toward the outflow tract in addition to reinforcing the vortices required to close the mitral valve. Following ejection, relaxation of the subendocardium is initiated near the apex and reaches the base at the end of isovolumic relaxation (11). As a result, the LV cavity initially relaxes near the apex, causing a brisk base-to-apex reversal of blood flow (5).

Because the counterdirectional orientations of fibers spiral into a vortex near the LV apex, the sequential transmural mechanical activity also results in rotational movement (which can be resolved by 2-dimensional [2-D] echocardiographic speckle tracking). A brief initial clockwise rotation of apex is seen during the isovolumic contraction period when the shortening occurs predominantly along the subendocardial fiber direction and is accompanied with stretching of the subepicardial fibers (Fig. 1). Subsequently, predominant subepicardial shortening results in prominent anticlockwise rotation during ejection (called "twist"). Dur-

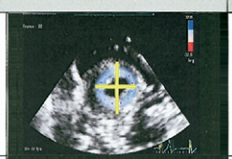
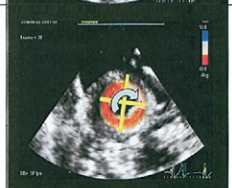
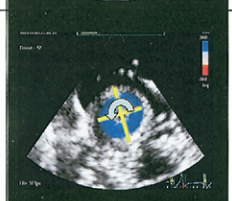
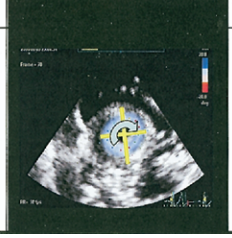
Phase	Subendocardium	Subepicardium	Apical Rotation		LV Cavitary Flow
Baseline	Relaxed	Relaxed	None		Concluding flow from atrial contraction
Isovolumic Contraction	Rapid apex-to-base electro-mechanical activation and initiation of shortening	Reciprocal stretching	Minimal clockwise twist (along subendocardial fiber direction)		Apex-to-base flow redirection without change in volume, flow acceleration accentuates vortex
Ejection	Apex-to-base contraction continues	Shortening starts and continues during ejection	Counter-clockwise twist (along subepicardial fiber direction)		Continued flow acceleration into aorta, flow from LV base redirected towards outflow
	Higher shortening strains at apex compared to base; descent of LV base				
Isovolumic and early Relaxation	Relaxation starts at apex and extends to base	Relaxation starts at base and extends to apex, apex still contracting	Clockwise untwisting (recoil and release of strain energy stored in deformed matrix)		Base-to-apex flow reversal, LV volume remains unchanged until the onset of early diastole
	subendocardial apex enlarges to initiate suction, subepicardial base relaxes to allow recoil. LV base ascends				

Figure 1 **Motion Sequences of the LV Wall and Intracavitary Flow Dynamics During Various Phases of the Cardiac Cycle**

LV = left ventricular.

ing this process, the subendocardial fibers continue shortening but are twisted across their helical direction, resulting in storage of systolic energy in a deformed matrix. During isovolumic and early relaxation, this stored energy is released in a brisk recoil, producing clockwise rotation (also called "untwist"). While the LV is untwisting, enlargement of the cavity near the apex is carefully modulated by a well-synchronized sequence of mechanical relaxation. The subendocardium relaxes and recoils first near the apex, and relaxation is completed near the base by the end of isovolumic relaxation. While the subepicardium relaxes near the base, allowing the onset of untwist, relaxation is completed near the apex at the onset of early diastole. This well-synchronized apical triple rhythm is the essence of the Waltz of the heart.

When Waltz and Vortex Go Wrong

The insights in mechanics provided by these studies have significant implications. It would appear that the Waltz and vortices can be exploited to provide superior pathophysiologic and diagnostic studies. One such application may include resynchronization therapy. Although QRS duration has been an inclusion criterion in all major trials, it has been shown that electrical dyssynchrony is not automatically associated with mechanical dyssynchrony and vice versa. Echocardiography is being widely used to identify dysssynchrony primarily because of high temporal resolution and ease of application. However, there is no gold standard for diagnosing dyssynchrony, so comparing the accuracy of the different echocardiographic parameters is not possible. By clarifying the relationship between the sequence of electric activation and its effects on LV mechanics and intracavitary rheology, one may get a better perspective on the most effective routing of blood flow in the failing heart. Recent investigations show that LV dyssynchrony is dynamic and changes with exertion (14). If LV mechanics and flow sequence are interlinked, does this change in synchrony reflect the changing blood flow pattern associated with exertion? Can correcting the sequence of blood flow be superior to adjusting the timing of regional contraction as a target for cardiac resynchronization?

Echocardiography may provide an excellent clinical tool to assess intracavitary flow dynamics. Digital particle-imaging velocimetry (PIV) is a well-established technique and is used as a gold standard for quantitative flow visualization in experimental fluid mechanics and industrial applications. The analogous use of contrast ultrasound for tracking the intracavitary flow by echo PIV provides an important advancement over 1-dimensional Doppler for noninvasive assessment of intracavitary hemodynamics (15). However, further validation of echo PIV would be required to integrate this into routine application. Vortical features of flow fields should be readily characterized on 2-D imaging (Kelvin's theorem). However, the development of high-temporal-resolution 3-dimensional (3-D) echocardiography and 3-D flow visualization with holographic techniques may provide a superior tracking and quantification of flow. Of note, 3-D flow visualization is currently available with magnetic resonance imaging (8), although at a temporal resolution that is lower than that required for tracking flow within the isovolumic period. As an alternative technique, assessment of the sequential contraction activity that produces the characteristic apical rotation triplet may provide a better measure of cardiac function. Early diastolic filling is contingent on adequate LV suction which is postulated to be associated with apical untwist. By 2-D speckle tracking velocity vector imaging, Li et al. (16) studied the apical rotation in normal subjects and those with LV hypertrophy and isolated diastolic dysfunction. The apical twist and untwist velocities were similar in normal patients. However, the untwist velocity was markedly decreased in LV hypertrophy. Maintenance of the delicacy of triple rhythm and coordinated vortices are integral to harmonious functions of the heart.

In aggregate, the foregoing studies are clarifying the interaction among LV anatomy, electric activation, and intracavitary flow. These findings, coupled with advances in noninvasive cardiac imaging technology, offer the potential to use cardiac contraction pattern and intracavitary flow as new descriptors of cardiac physiology and pathology. The observations of previous centuries may thereby be translated into valuable clinical tools (13).

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